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THE MORBID CHANGES IN THE PUERPERAL ENDOMETRIUM DUE TO LOCAL INFECTION, AND THEIR RELATION TO GENERAL SYMPTOMS.*

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The modern treatment of early infection in the puerperal state illustrates the theory of its origin. The point of attack has been proved to be the endometrium by the efficacy of treatment when applied to the cavity of the uterus in relieving the general symptoms.

According to the classification of Bumm (1) there exist two primary forms of puerperal endometritis: putrid and septic. Inasmuch as the developed manifestations of septic forms of endometritis demand surgical intervention and are more properly considered in the surgical discussion of the subject, we shall confine our attention to the origin of septic infection in the endometrium and to the local morbid changes due to putrid infection.

Septic endometritis occurs in two forms: First, a localized septic process in which a granulating zone, shutting off the necrotic endometrium and preventing the invasion of germs, is present. The uterine lymphatics are not actively involved. The placental site, as in the putrid form, is most markedly affected. Secondly, a septic endometritis, accompanied by a general infection. Bumm has studied five cases belonging to this class, and has found in three instances that infection has occurred by invasion through the lymphatic system, and in two instances along the course of the veins. In the first set of cases examination shows the placental site free from micro-organisms and thrombi; the lymphatics generally exhibit accumulations of streptococci and staphylococci which penetrate directly to the peritonæum. In the second set of cases only the smaller lymphatic branches surrounding the sinuses are marked by colonies of cocci; from these isolated lymphatic channels the accumulations of cocci penetrate to the larger lymphatics underlying the peritoneal covering of the uterus.

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The decidua is disorganized and infiltrated with a fibrous exudate presenting a diphtheritic appearance. In this class of cases, as well as in that just described, the granulation zone is absent. This fact has evidently an important bearing upon the function of such a zone of demarcation, in combating the progress of micro-organisms into the underlying tissues.

Although putrefactive germs may be present in all forms of septic endometritis they do not predominate, consequently fœtid lochial discharge, the sign of necrotic decidual tissue and disorganized thrombi, usually do not accompany the more active septic processes.

The active cause of puerperal septic infection attacking the endometrium is the presence of the pyogenic cocci (so called), namely, *Streptococcus pyogenes aureus*, the *Staphylococcus pyogenes aureus* and *albus* and the *Streptococcus erysipelatus.*

These germs attack the endometrium upon the placental site or at points of denudation the result of lacerations about the cervical canal; abrasions and contusions of the vulva and vagina are less likely to communicate the infecting material to the general circulation on account of the less active lymphatic circulation from these parts owing to the comparatively moderate development during pregnancy of the lymphatic vessels accompanying the pudic and uterine vessels, in distinction to the active development of those corresponding to the ovarian vessels running to and from the fundus of the uterus.

The causes which predispose to local infection, inasmuch as they act as preparatory influences in their effects upon the endometrium, are, first, the earlier local changes due to former endometritis (notably the presence of infecting cocci in a dormant state); second, the constitutional character of the gravida, determining the power of resistance of her tissues to infecting elements; third, the degree of uterine contraction; fourth, puerperal hæmorrhage; fifth, the presence of gonococci and the resulting local changes due to them.

As to symptoms we find by examination in localized infection abdominal tenderness and imperfect involution, the uterine tumor presenting an abnormally yielding consistence. Internal examination by means of the speculum reveals a cervical portion covered with patches of discolored deposit (diphtheritic) alternating with areas of necrosis and granulation. The vagina is reddened, glazed and sensitive to the touch. Among the general symptoms we have those of a mild irritative fever often marked by roseolous eruptions over the chest and neck, together with the presence of fœtid lochia. In the acute form

of infection with active lymphatic absorption there are present either the symptoms dependent upon peritonitis or those due to the active absorption of septic germs and their products, including pyæmia among these symptoms.

In discussing the putrid form of endometritis it will be better to use the term necrotic endometritis, as the germs of putrefaction are only active in an environment of unorganized tissue. We shall also speak of saprophytes as mycotic organisms in contradistinction to their parasitic classification.

The general symptoms accompanying necrotic endometritis are those which are classed under the head of sapræmia. A careful study of the pathology and ætiology of sapræmia will throw important light upon the development of these symptoms. In the broadest sense the cause of necrotic endometritis is the retention of putrid material within the uterus. This may be in the form of decidual *débris* or of putrid fœtus; it may be the result of sloughing following pressure, or of the previous condition of the endometrium, as in carcinoma or the various forms of non-puerperal endometritis. As to the pathology we have to deal with, first, a general necrotic condition of the endometrium, and secondly, a condition of necrosis localized in the placental site. The remnants of decidua retained within the uterus, the placental *débris* and the thrombi which project from the placental site are prone to putrefactive changes in the presence of saprophytes. Histologically we detect a necrosis of the epithelium and basement membrane, the necrotic tissue being cut off by an inflammatory zone of small-celled infiltration extending into the muscular layer and choking of the lymph spaces. In other words this zone of reaction acts as a barrier against the products of putrefactive change; as a result of this we find that of all forms of puerperal endometritis this form is most distinctly localized in its effect. The only point where this protection is weakened is at the placental site. Invasion of the thrombi occurs here owing to the want of organization of the thrombotic tissue. At this point the sinuses become filled with necrotic material and the lymphatics surrounding them act as channels for the absorption of the bacterial and degenerative products of putrefaction. In this instance it is incorrect to speak of the process as an endometritis, as the muscular layer is involved in the process and the condition is better described as a metritis. A less frequent mode of invasion of putrid germs producing metritis is that by which the microorganisms are absorbed from necrotic areas in the lower segment of the uterus and cervix the result of lacerations during parturition.

Unfortunately the bacteriology of putrid infection has not been worked out to the same extent to which the study of septic infection has been developed. We are led by recent researches however to include many of the so-called non-pyogenic germs, among the pus-producing organisms. For instance within the last few years the specific germs, such as, the bacillus typhosis, and the *Diplococcus pneumoniæ*, as well as the *Bacterium coli communis*, (2) also the saprophytic germs, the *Micrococcus tetragenus* and the *Bacillus pyogenes fœtidus*, (3) have been classed as pyogenic organisms. We are further confronted by the fact that certain chemical substances under peculiar circumstances are capable of producing suppurative processes, as proved in the case of putrescin and cadaverin.

Associated with this pathological condition we have the symptoms characteristic of two degrees of putrid infection or sapræmia, the mild and the grave form. In each case the morbid manifestations outside of the local condition are usually those due to a toxæmia, although, as noted above, the products of putrid changes may be capable of producing suppuration as in septic infection, so that at the present point to which recent investigation has led us we are not able to divide the sapræmic and septic infections distinctly as to their cause. The pathological changes, both local and general are certainly distinct, but the chemical and bacterial products of sapræmia, on the one hand, are capable of producing suppurative changes analogous to those found in septic infection, while on the other hand the mild infections characterized by parametritic infiltrations and local accumulations of the pyogenic cocci are sometimes due to a mixed infection (putrid and septic) in which the irritative disturbance depends as much upon the chemical products of putrefaction as upon the presence of septic germs.

The grave septic conditions are often unaccompanied by symptoms of local putrid changes, such as fœtid lochia and the discharge of necrotic decidua. In this case the lymphatics have actively absorbed the septic products and the local reactionary changes (small-celled infiltration and the formation of a dense inflammatory zone) have been moderate. In fact it appears to be the rule that where we take into account the seat of infection and the systemic action the result of infection we find the latter more pronounced in cases where the local changes have been of a mild nature. It has been suggested that the preponderance of the general over the local changes depends not so much upon the products of the bacterial elements set free at the point of infection, namely the ptomaines, as

upon the death of the plasma contents of the bacterial cell itself the result of the combative qualities of the local cells (4). Abbott however states: "We are not as yet in a position to say definitely to which of these influences the death of the tissue is due, or, indeed, that it is, when occurring spontaneously, the result of the action of the one to the exclusion of the other." He believes that the local necrosis occurring in suppuration is dependent upon both the poisonous products of growth of the living bacteria and the poisonous activities of the proteid constituents of the dead and disintegrating bacteria.

In conclusion it is important to note that putrid changes at the seat of infection have an important bearing upon the spread of infection, rendering the septic germs more active than they would be in attacking tissues unaffected by necrotic and putrefactive changes. In other words, the saprophytes, with or without causing symptoms of intoxication, may render the individual receiving them more susceptible to infection from other bacteria (5, 6). This fact bears upon the cases in which putrid changes in the endometrium accompany the grave symptoms of septic infection, such as those due to metrolymphangitis and peritonitis.

References.

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